

## DISSEMINATED AMERICAN CUTANEOUS LEISHMANIASIS IN A PATIENT WITH AIDS

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American cutaneous leishmaniasis has been found in almost all States and Territories in Brasil (Deane & Grimaldi, 1985, In: K.P. Chang & R.S. Bray, *Leishmaniasis*). Two complex of parasites, *Leishmania braziliensis* and *L. mexicana* were defined by Lainson & Shaw (1972, *Brit. Med. Bull.*, 28: 44-48) based on morphology and behavior in humans, hamsters, cultures and sandflies. The following species or subspecies from humans have been identified biologically and biochemically and classified by zimodemes, schizodemes and serodemes (monoclonal antibodies) in this country: *L. braziliensis braziliensis*, *L. braziliensis guyanensis* and *L. mexicana amazonensis* (Miles et al., 1981, *Trans.R.Soc.Trop.Med.Hyg.*, 75: 524 - 529; Lopes et al., 1984, *J. Parasitol.*, 70: 89 - 98; Pacheco, 1985, Thesis, Instituto Oswaldo Cruz, 143p. and Grimaldi et al., 1987, *Am.J.Trop.Med.*, 36: 270 - 287). Two other parasites differing from both by the same methods have been also described (Grimaldi et al., 1983, X Reun. Pesq. Bas. D. Chagas, Abs. 128 and Momen et al., 1985, *Am.J.Trop.Med.Hyg.*, 34: 1076 - 1084).

The *L. braziliensis* complex in man, in general, causes few lesions in number, the parasites are smaller and scanty, grow poorly in culture media, but the lesions can be extensive and disfiguring with a high risk of mucosal invasion in cases of *L. braziliensis braziliensis*. No case of diffuse cutaneous leishmaniasis has been shown to be produced by subspecies of *L. braziliensis* complex. Conversely in the *L. mexicana* complex the parasites are larger, more numerous in the lesions, grow easily in culture media, produce few ulcerated cutaneous lesions which may be self-healing, without mucosal invasion. Several cases of diffuse cutaneous leishmaniasis have been proved to be produced by the *L. mexicana* complex.

Until recently there was no description of the effect of the acquired immunodeficiency syndrome in cases of American cutaneous leishmaniasis (Coura, 1987, *J. Bras. Med.*, 53-54).

**Case report** - In January, 1987, one of the authors (JRC) was called to see the patient R. T.F., male, 57 years old, married, living with his family in good conditions in the south section

of Rio de Janeiro city. The patient was "feeling sick" in the last two months. He was feverish, weary, anorectic, easily fatigable and irritable. He had a profuse sweating at night and some weight loss in the last days, when he could not also sleep well because of a sensation of obstruction on his nose that impaired the normal respiration. There was no diarrhea or other symptoms of localized disease in any organ or system.

The clinical examination showed numerous skin erythematous papules or nodules, non-ulcerated in the face, thorax and lower limbs, with a variable diameter, from one to three centimeters, with a redish inflammatory base. The nasal mucosa was congested but there was no ulceration, however, in the soft palate there was a small ulcer of approximately one centimeter in diameter, with signs of acute inflammation. There was no enlargement of the lymph nodes and the spleen and liver were unpalpable. The clinical examination of the hearth, lung and nervous system was normal.

The patient had several years ago an unexplained "myositis" of several groups of muscles in the upper and lower limbs, thorax and abdomen, with some muscle necrosis, which led to surgical ablation. This syndrome which lasted for a few years, was self-cured without any definitive explanation. Because of this it was thought that the present disease could have some relationship with his past syndrome. On the other hand the patient had spent some time in the last two years in the Jari Project, in the Amazon region. Although leishmaniasis could not explain all the feature of his clinical manifestations it should be one of the hypothesis for diagnosis. Therefore a deep biopsy in one lesion was made to try to confirm the diagnosis. Unfortunately the skin test for leishmaniasis could not be done at that time.

Several laboratory tests were performed including lymphocyte count, OKT4 and OKT8 differential counting and level of immunoglobulins and complement. The global number of T lymphocytes was 838 per mm<sup>3</sup>, 23% of T helper (OKT4) and 37% of T supressor (OKT8), with a ratio of T4/T8 of 0.6. The proportions of the total lymphocytes were 51% of T, 19% of B and 30% of undifferentiated lym-

phocytes. The dosage of IgG was 908 mg/DL, IgA 485 and IgM 138/DL. The complement C3 was 98 mg/DL and C4 86 mg/DL. The histopathology of the skin biopsy showed a lymphohistiocytic infiltrate, plasma cells and numerous amastigote forms of leishmania within the macrophages. The granulomas were scanty and not very consistent. The monoclonal antibody specific for the amastigote stage of *L. mexicana amazonensis* in frozen tissue sections embedded with immunoperoxidase (Grimaldi et al., in press) did not recognize the parasites, therefore we concluded that the parasite involved in the lesions should be the *L. braziliensis braziliensis* which can invade the mucosa.

Although there was no clinical or epidemiological indication that the patient was in the risk group for AIDS (he was married, heterosexual, did not use blood or blood products in the past and was not a drug abuser), because of the inversion of T4/T8 ratio and the result of the skin biopsy, we performed a serology for HTLV/III which was strongly positive in ELISA and immunofluorescence. Before we had the final result of the serology the patient had a pneumonia by *Pneumocystis carinii* and died one week later due to respiratory insufficiency. He was an international consultant for administration and had several travels all over

the world and we suspect he acquired AIDS in heterosexual contact with somebody in the risk group.

*Comments* – As mentioned before we could not find in the medical literature any other description of American cutaneous leishmaniasis disseminated or modified by concomitant infection with AIDS virus; however, the immunosuppression caused by that kind of virus logically should affect the cellular immunity in humans elicited by ACL. On the other hand the case reported is very similar to diffuse cutaneous leishmaniasis, described as an anaergic type of leishmaniasis usually caused by the *L. mexicana* complex. In our reported case we could exclude this type of parasite using one specific monoclonal antibody to the amastigote stage of *L. mexicana amazonensis* in frozen tissue sections embedded with immunoperoxidase.

*Acknowledgements* – We wish to thank Dr. Leonidas M. Deane, Department of Entomology, Oswaldo Cruz Institute for his critical reading of the manuscript and pertinent suggestions and Dr. Antonio Marques, Department of Pathology, Medical School, Federal University of Rio de Janeiro, who did the skin biopsy in the patient and made the preliminary histopathologic diagnosis of leishmaniasis.